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VALUE OF DYSLIPIDEMIA IN THE PATHOGENESIS OF OSTEOARTHROSIS IN CONJUNCTION WITH ESSENTIAL HYPERTENSION

Purpose. To study the influence of dyslipidemia on the articular syndrome manifestations and circadian rhythm of blood pressure (BP) in patients with osteoarthritis (OA) in combination with essential hypertension (EH). Objects and methods. In 120 patients with OA in combination with EH and in 60 EH patients (control group) blood lipids were determined, the content of IL-1β, tumorigenic-necrotic factor-α, C-reactive protein, algofunctional Lequesne index, C-terminal telopeptides, transport of calcium between the skeleton and blood, endothelial function, circadian rhythm of blood pressure. Spine X-ray densitometry was performed. Results. In OA patients in combination with EH in greater stage ($p<0,05$), than in the control group there were expressed Dyslipidemia, endothelial dysfunction, increased levels of markers of inflammation, bone resorption, slow transport of calcium between the extracellular and bone deterioration sectors and circadian blood pressure. A moderate correlation has been established between the levels of cholesterol (CS), low density lipoprotein (LDL), and endothelial dysfunction degree ($r=0,543$; $p<0,05$), calcium transport intensity ($r=-0,557$; $p<0,05$) and algofunctional Lequesne index ($r=+0,551$; $p<0,05$). Problems of calcium transport are most manifested in patients with circadian rhythm of blood pressure - night-peakers and high (>3.80 mmol/L) LDL cholesterol. Conclusion. Dyslipidemia is one of the common pathogenetic mechanisms of OA and EH.

INTRODUCTION

Osteoarthritis (OA) and essential hypertension (EH) are socially significant diseases that require development and implementation of effective therapeutic and diagnostic procedures. According to the NSC "Institute of Cardiology named after Academician N.D. Strazhesko of NAMS of Ukraine," in Ukraine EH affects about 44% of the adult population, the prevalence of OA is 3172.6 per 100 thousand of population, and the incidence - 607.3 cases per year. The high prevalence of EH and its associated risk of injury to vital organs determine its place among the key causes of disability and mortality (Kovalenko V.M. , Kornatsky V.M. (Eds.), 2008). OA is also one of the leading causes of functional impairment and disability in the adult population (Kovalenko V.N., Bortkevych O.P., 2005). However, in recent years more and more often there is evidence that patients with OA have a higher risk of cardiovascular disease and mortality compared with the general population. It has been established that the OA of hands joints associated with increased cardiovascular mortality in men (Zoler ML, 2009; Mendel O. I. et al., 2010; Eustise S., 2010).

Virtually there are no individuals with OA without comorbid medical illnesses. Typically, a patient with OA over the age of 50 years coexist more than 5 diseases and OA is not a simple addition to other diseases. OA is most often combined with EH. Publications analysis indicates that the combination with EH is observed in 48-65% of patients with OA in the population. EH and OA mutually burden treatment of each other and complicate the selection of optimal treatment (Gislason G.H. et al., 2009; Zoler M.L., 2009). Both diseases lead to loss of quality and duration of life. Despite significant progress in recent years in the study of EH and OA, questions remain about how to activate their common pathogenetic mechanisms and characteristics of therapy in case of their combined flow. For example, it was found that the use of non-steroidal anti-inflammatory drugs (NSAIDs), which provides pronounced anti-inflammatory and analgesic effect in OA in patients with a history of heart disease, by 10 times increases the likelihood of hospitalization for heart failure and leads to destabilization and progression of EH. An increased risk of cardiovascular events - heart attack, stroke and sudden cardiac death - can be considered as a class-specific side effect for all NSAIDs. In addition, NSAIDs may reduce the effectiveness of antihypertensive drugs, especially those which effect is mediated through the renin-angiotensin system (beta-adrenergic blockers, angiotensin-converting enzyme (ACE) inhibitors, diuretics, angiotensin II receptor antagonists), inhibition of which is accompanied by a decrease in the synthesis of prostaglandins and endothelial and other vasodilators (Gislason G.H. et al., 2009; Trelle S. et al., 2011).

In the third study of National Health and Nutrition of the population in the United States in 1988-1994, with the participation of 77,714 people (of which 975 had OA), OA patients were more likely than the general population to show dyslipidemia, elevated triglyceride (TG) - 47% versus 32% and hypertension - in 75% of cases, compared with 38 % (Rosenberg L. et al., 1995). These results are not accidental: common pathogenetic mechanisms of these two pathologies are assumed, which are based on endothelial dysfunction (Lupinskaya Z.A. et al., 2008; Zwinger S.M., Govorin A.V., 2009). The development of degenerative changes in the vascular wall and the articular cartilage and progression of OA and EH may be causes by lipid metabolism problems, which are common pathogenetic mechanism of these diseases (Masuko K. et al., 2009; Aspden R.M., Scheven B.A., 2010). An important factor in violation of the regulation of vascular tone is endothelial dysfunction, which develops due to a lack of endothelial synthesis of NO, which provides anti-inflammatory, anti-proliferative and anti-adhesive properties of the endothelium. Under dyslipidemia, oxidized low density lipoproteins (LDL) reduce the activity of endothelial NO-synthase (NOS) and the bioavailability of NO (Rubbo H., Freeman B.A., 2008). Therefore, analysis of the factors of endothelial dysfunction in patients with a combination of OA and EH is well founded. In response to the damaging effects of excess blood lipids endothelium responds to increased synthesis of vasoconstrictor and vasodilator inadequate synthesis. Chronic activation of the immune inflammation also contributes to this. Adhesion molecules appear on the membrane of endothelial, ensuring penetration in the vascular wall of T-lymphocytes and macrophages. Inflammatory blood cells produce interleukin (IL) -1 and -6, stimulating synthesis of C-reactive protein (CRP), fibrinogen and angiotensinogen in the liver (Boos C.J., Lip G.Y.H., 2006; Braunwald E., 2011).

In the articular cartilage activated CRP macrophage-like synoviocytes and white blood cells infiltrating the synovial membrane of the joint, produce cytokines, free radicals, etc., providing

resorption by extracellular matrix by chondrocytes and cartilage erosion, which increases the load on the subchondral bone, where microfractures occur (Goldring S.R., Goldring M.B., 2004; Bondeson J. et al., 2010). It is known that if OA is present, the frequency of osteoporosis (OP) is 28.9% in females and 20% in males, osteopenia - 52.9 and 38.8% respectively (Goldring S.R., 2009). In patients with OA loss of bone mineral component is accelerated, and the decrease of bone mineral density (BMD) results in a more pronounced degradation of cartilage and joint damage progression (Buckland-Wright C., 2004; Dore D., Quinn S., 2010; Grynpas M.D., 2010).

In patients with EH, infringements of calcium metabolism were discovered, characterized by a tendency to a deficiency of extracellular calcium and activation of the synthesis of parathyroid hormone, which accelerates the resorptive processes in the bone tissue (Kolomiyets V.V. et al., 2009). Peak bone mass is determined by the rate of bone formation and resorption of it, which is closely related to the content of calcium in the extracellular space of the body (Benevolenskaya L.I., 2005).

Disorders of calcium exchange are involved in the development of not only of osteopenic syndrome, but EH as well, coronary heart disease, chronic heart failure. Reduced bone mineral density and fractures are treated even as independent risk factors for cardiovascular disease (Zoler M.L., 2009; Eustise C., 2010). Thus, endothelial dysfunction is accompanied not only by the rise in blood pressure (BP), but by deterioration of intraosseous hemodynamics in subchondral bone tissue, which leads to dystrophic changes in articular cartilage and progression of OA.

The purpose of the study is to analyze the significance of dyslipidemia in endothelial dysfunction in a state of resorption and bone mineralization, circadian blood pressure profile and articular manifestations of the syndrome in patients with OA in combination with EH.

MATERIALS AND METHODS OF RESEARCH

In 120 patients with OA of knee joints of I-II radiographic stage acc. to Kellgrenn (diagnosis of OA is set based on the criteria of the Association of Rheumatology of Ukraine) aged 52 to 75 years, on average - 64,7 ± 1,6 years (OA disease duration was on average 9,1 ± 1,7 years), in combination with EH of II stage, the duration of which amounted to an average of 10,9 ± 1,7 years (main group) and 60 patients with EH stage II (the 1st control group which did not differ from the main one in gender, age (49-76 years, mean age - 62,3 ± 1,8 years), duration (10,1 ± 1,5 years) and EH stage) blood lipid range was determined by colorimetric method: total cholesterol (TC), high density lipoprotein (HDL) cholesterol, triglycerides, and (in 38 patients of the main group and in 36 patients of control group), - the content of IL-1β, tumor necrotic factor (TNF)-α using the set "Ukrmedservis" (Donetsk, Ukraine) and CRP by a set of "DRG International Inc." (USA). Joint function was assessed using the algofunctional Lequesne index. The degree of bone resorption was tested by quantitative determination of urinary C-terminal telopeptides using ELISA kit "CrossLaps^{TM} ELISA" at spectrophotometer "Sanofi Pasteur PR2100". Assessment of the structural and functional state of bone densitometry was performed by X-ray of the lumbar spine with the machine "Lunar DPX". Osteoporosis is diagnosed under the condition of deviation of the BMD> -2,5 SD from the norm; osteopenia – at a deviation of indicators of BMD from -1 to -2,5 SD.

During 3-4 days and during the survey, the standard diet No. 10 was recommended (acc. to M.I. Pevzner) with which the organism accepts about 16.5 mmol of calcium per day. One day before the survey, it was recommended to exclude dairy products from the diet, for the daily calcium intake not exceed 10 mmol. The concentration of calcium in serum and urine were determined by the set of "Filisit-Diagnostika" at "Specord" spectrophotometer.

The state of bone calcium depot was characterized indirectly by assessing intake of calcium from bone into the blood plasma. The method is based on the assumption that the level of calcium coming in blood - after a meal break at night – is due to the effect of calcium-regulating hormones on kidneys and bones and is dependent on functional state of these organs. The concentration of calcium in the blood serum was checked 18h after the meal and in urine collected in the morning on an empty stomach 120 minutes before. Proceeding from the idea that in normal tubular reabsorption of calcium, the level of calciemia at fasting depends primarily on calcium entry into the bloodstream from the skeleton, the algebraic difference between the deflection of the concentration of calcium in the blood serum of patients on the calcium concentration in healthy individuals and the amount of calcium coming in the blood from kidneys by renal tubular reabsorption was calculated, which coincides with the value of calcium coming from the extracellular fluid into bone tissue. 24 hours BP monitoring was performed using "BAT41-2" device. Endothelial function was studied using sonographic study of dilatation of brachial artery (BA) in the reactive hyperemia (RH) at "ULTIMA PA" device, and measurement of nitrite (NOx) in plasma with Griess reagent in a spectrophotometer "Specord 200 PC".

Control group 2 included 20 healthy persons aged 43-72 years, mean age - 60,1 ± 1,9 years.

The study excluded patients with diabetes, heart failure of III-IV level, chronic diseases of kidneys and digestive system.

The results are processed statistically using the software package "Microsoft Excel" and the program "Biostatistics 4.3" (USA).

RESULTS AND DISCUSSION

For patients of the main group, the algofunctional Lequesne index used to evaluate the functionality of joints made 7,0-15 points, on average - 9,5±0,7 points, indicating a moderate severity of articular syndrome.

The results of the survey of the main group and the 1st control group indicate they have endothelial dysfunction. This is confirmed by the lack of (<10%) in endothelium-dependent vasodilation PA against WP, but its average value at the main group (+6,8 ± 0,5%) was significantly ($p<0,05$) lower than that of a control group of patients (+8,2±0,6%). Concentration of stable metabolites NOx in blood plasma of patients of the main group (34,4±1,5 mmol/l) was significantly ($p <0,05$) lower than that of a control group of patients (46,2±2,3 mmol/l). Normally, NO is metabolized to nitrites and nitrates. Its small part interacts with superoxide to form peroxynitrite. With such a pathology, as OA and EH, when production of free radicals, including superoxide anion, is increased, most of the NO is destroyed, resulting in reduced its bioavailability and increase the degree of dysfunction of endothelium. In patients with OA in combination with EH, creation by endothelium of vasoconstrictor factors is maintained or increased in response to the stressor effects

of joint syndrome with pain and limited function of large joints, while the resistance is provided by NO, performing the role of a "moderator" of local and systemic vascular tone. One of the causes of endothelial dysfunction may be hypercholesterolemia. It is known that LDL cholesterol leads to increased synthesis of caveolin-1, which inhibits the synthesis of NO by inactivation of endothelial NOS, the shortage of which leads to the formation of free radicals. LDL and their oxidized forms cause a cascade of pathophysiological reactions in the beginning at the cellular level, and then – at multiple organ one, with damage of aimed organs and development of cardiovascular complications. LDL cholesterol can reduce the sensitivity of receptors of the vascular wall to the action of antihypertensive drugs (Yang Z., Ming X.-F., 2005).

Total cholesterol levels in both groups deviated in the same range: 4,69-7,5 mmol/L in the main group and 4,5-7,6 mmol/L - in the 1st control group, the average total cholesterol levels in both groups (main - 5,97±0,24 mmol/l, control - 5,83±0,21 mmol/l) also did not differ significantly. However, it is clear that not only the average, but the minimum content of total cholesterol was above the recommended level for those with the presence of at least one risk factor (EH) - <3.0 mmol/l. In both groups, there was an increase in LDL-C, making an average of 3,78±0,07 mmol/L in the main group and 3,53 ± 0,10 mmol/l - in the 1st control group, that is, in patients with EH combined with OA it was higher ($p < 0,05$). HDL cholesterol in the main group (1,09±0,06 mmol/L) is lower ($p < 0,05$), than in the control group (1,31 ± 0,08 mmol/l). Atherogenic factor in patients of the main group (4,43±0,19) was significantly higher ($p<0,05$) than in patients with average control group (3,63±0,23), which is also beyond the upper limit of the maximum normal values - 3.5.

Excess lipids of blood causes damage of endothelial, responding with synthesis of vasoconstrictors, and activation of the renin-angiotensin and sympathoadrenal systems (Rebrov A.P. Kharitonov, I.A., 2007). Between the levels of LDL cholesterol and increase the diameter of the PA values against WP there is a correlation of moderate distress ($r=0,543$; $p<0,05$). Angiotensin II is the primary antagonist of NO, not only inhibiting its synthesis, but also making the already synthesized NO in toxic peroxynitrite, which destroys the endothelial cells and oxidizes LDL, which are harmful to the body. Angiotensin II also increases the expression of endothelial receptors for LDL cholesterol and uptake of LDL cholesterol by endothelial cells (Yang Z., Ming X.-F., 2005).

These processes enable the chronic immune inflammation and contribute to the development and progression of a number of systemic pathologies (EH, atherosclerosis, coronary heart disease, including heart attack, stroke). Under the influence of oxidized LDL, the adhesion of leukocytes to the endothelium increases, the synthesis of IL-1 monocytes is induced, the expression of a large number of growth factors, cytokines, TNF-α (Tedgui A., Mallat Z., 2006).

Levels of IL-1β in patients of the main group (26,7± 0,4 pg/ml) significantly exceeded ($p<0,05$) the level of the control group participants (20,2 ± 0,3 pg/ml) and healthy individuals (12,1±0,1 pg/ml). Serum TNF-α in patients of the main group was 16,5 ± 0,2 pg/ml, 2 times exceeding ($p<0,05$) its content in healthy individuals (8,1±0,1 pg/ml) and 1.3 times ($p < 0,05$) in a control group of patients (12,9 ± 0,1 pg/ml). These data indicate systemic inflammation, pronounced in patients with a combination of OA and EH. It is proved that low-intensity systemic inflammation plays an important role in the pathogenesis of cardiovascular disease, including ethylene glycol, and increases the risk of vascular complications, which correlates with the content of serum CRP. The

results of the 8-year study (The Black Woman's Health Study) showed that survival with combined LDL-C <3.4 mmol/l and CRP>2 mg/L was worse even than in the case of a higher LDL cholesterol and normal CRP levels (Rosenberg L. et al., 1995). Therefore, CRP is considered as a marker of increased cardiovascular risk. Patients of the main group had CRP 2,64±0,05 pg/ml significantly higher than the level in patients from the control group (2,27 ± 0,04 pg/ml). Oxidized LDL and inflammatory cytokines increase the permeability of the endothelium, leading to the proliferation of muscle cells in the vascular wall thickening of the intima-media by 2-3 times, which increases the stiffness of the arteries, and helps development of refractory to treatment of EH (Ferroni P., Basili S., 2006).

When ABPM in two study groups, no significant difference between the average daily blood pressure were observed. Patients in each of the two groups were divided by the degree of nocturnal BP reduction, the inadequacy of which is associated with the highest incidence of cardiovascular complications. Daily profiles of blood pressure type "non-dipper" and "night-peaker" were observed in ⅔ of patients with OA and EH, and slightly less than half of the patients with EH. The examined patients had increased variability of systolic blood pressure (VMSBP) (> 15 mm Hg) in 40% of cases in the main group and in 20% of cases in patients from the control group. Average VMSBP in OA patients in combination with EH (15,6 ± 0,8 mm Hg) is significantly higher ($p<0,05$), than in patients with EH (12,3 ± 0,5 mm Hg). The VMSBP value in patients of the main group exceeds the maximum permissible level, which is 15 mm Hg. It is obvious that during the day blood pressure significantly changes under the influence of various internal and external stress factors, which may include joint and pain with movement. Arthritic pain by adrenergic stimulation increases sympathetic activity. Between algofunctional articular Lequesne index and the severity of changes in BP profile, moderately pronounced correlation has been established ($r=+0,487$; $p<0,05$).

This reduction of an active NO leads to influences of oxidized LDL on endothelial cells, and increased synthesis of peroxynitrite also contributes to the destruction of articular cartilage, as in articular cartilage there are analogous processes: activated DRR synoviocytes and macrophage leukocytes infiltrating the synovium joint, produce IL-1, TNF-α, free radicals that cause resorption of the extracellular matrix by chondrocytes (Masuko K. et al., 2009). Matrix metalloproteinases and agrecinazes destroy collagen of type II and aggrecan, they are substituted with collagen I, III, and X-type and decorin. At this stage OA clinically manifests. Articular cartilage slowly undergoes erosion, which significantly increases the load on the subchondral bone, and leads to the formation of a large number of microfractures in the subchondral bone. Compensatory sclerosis of subchondral bone is developed, the rigidity of the bone tissue increases, which in turn contributes to further destruction of the cartilage tissue. Blockade of IL-1 effectively prevents destruction of the articular cartilage. Blockade of TNF-α leads only to reducing of inflammation in joint tissues. It is proved that the use of statins effectively normalizes endothelial NOS, which has an indirect effect such as anti-inflammatory in the vascular wall and in the articular cartilage and subchondral bone. In the Rotterdam study showed reduction of X-ray changes in the knee joints at OA under the influence of statins. However, clinical and functional manifestations of joint syndrome have not been assessed (Hofman A. et al., 2009). Direct damaging effects of lipid metabolism in the articular cartilage is assumed (Clockaerts S. et al., 2010). Between the levels of LDL cholesterol and algofunctional

Lequesne index in examined patients of the main group there was found a direct correlation ($r=+0,551$; $p<0,05$).

OA is also characterized by an imbalance between the anabolic and catabolic processes in cartilage and subchondral bone, resulting in accelerated loss of the mineral component of bone. The acceleration of bone resorption is evidenced by revealed in the main group the higher ($p<0.05$) concentrations of the bone resorption marker of C-terminal telopeptides (252,5±16,1 mg/mmol of creatinine) than in patients from the control group (189±14,8 mg/mmol of creatinine). Between LDL cholesterol levels and indicators of C-peptide concentrations in the urine moderate positive correlation was identified ($r=-0,44$; $p<0,05$). The result of the accelerated bone resorption is its demineralization.

In OA patients in combination with EH of the main group (66 persons examined) osteoporosis (18 patients) and osteopenia (31 patients) revealed significantly ($p<0,05$) more frequently (respectively in 27.3%, and 47.9% of cases) than in the control group (36 people surveyed), in which osteoporosis was detected in 5 (13.9%) patients, and osteopenia - in 12 (33.3%) patients. Normal BMD, age-adequate, were observed in 19 people in the control group (52.8% - nearly half of all surveyed), and only 17 patients of the main group (25.7% of cases - about ¼ of surveyed).

The average value of BMD in patients of the control group was 1,09± 0,04 g/cm ^{2}. The average value of this indicator in OA patients in combination with EH of the core group was significantly ($p<0.05$) lower - 0,90±0,02 g/cm^2. Thus, bone health shows more pronounced changes in its system in patients with a combination of OA and EH than in patients with EH without OA.

Since concentration of calcium in the blood serum is one of the key indicators of homeostasis, its maintaining at a constant level is provided by the whole regulatory system that includes a hormonal link (parathyroid hormone, calcitonin and vitamin $D_{3)}$ and executive organs (bones, kidneys, intestine, intracellular sector). However, given the reduction in BMD, it is reasonably to imagine that the persistence calcemia is achieved through tension of regulation system and at the cost of failure of this element in other less important sectors, one of which is bone tissue.

In healthy persons, the intensity of calcium transportation between kidneys and extracellular fluid (+0,108 ± 0,010 mmol/l) is in dynamic equilibrium with the intensity of its transportation between extracellular fluid and bone depot (-0,108 ± 0,009 mmol/l). In patients with EH, intensity of mineral transportation between extracellular and bone sectors is significantly reduced (-0,076 ± 0,007 mmol/l; $p<0,05$), while calcium transportation between kidneys and extracellular fluid differs less (+1,049 ± 0,015). But even to the greater extent calcium transportation between extracellular and bone sectors is slowed (-0,047 ± 0,007 mmol/l; $p<0,05$) in EH patients in combination with OA, which is confirmed by predominance of calcium resorption from bone tissue. Analysis of individual indicators of calcium transport showed even its opposite direction in 31 (25.8%) patients in combination of EH with OA. In 33 (27.5%) patients, calcium transportation between skeleton and extracellular fluid was below the lower level of volatility of this indicator in the group of healthy individuals. Between the levels of LDL cholesterol and calcium transport intensity there was found a significant negative correlation ($r = -0,557$; $p<0,05$). Slow movement of calcium between the bone and extracellular fluid is directed on the one hand, to a retention of calcium in bone, and on the other hand - to maintaining normal levels of calcemia under conditions of low calcium income from the

intestine or its excess loss be kidneys. As a result, for patients with EH in combination OA, equilibrium is achieved by reducing bone mineralization. Reduced bone mineral density leads to cartilage degradation. Thus, remodelling of bone tissue reflects the abnormal calcium metabolism, contributing to the development of OA as well as EH. So, between the level of urine C-peptides and algofunctional Lequesne index a significant, although a weak negative correlation has been found ($r=-0,31$; $p<0.05$).

Calcium leaving bones is excreted by kidneys. There is a lack of calcium in extracellular fluid, which in turn leads to stimulation of parathyroid hormone that promotes the accumulation of calcium in the smooth muscle cells of blood vessels and improves their tone and blood pressure. In most, calcium transportation between extracellular and bone sectors is slowed in the main group of patients with circadian rhythm of blood pressure - night-peakers, which is associated with a greater risk of cardiovascular complications (-0,043 ± 0,003 mmol/l; in patients with other blood pressure rhythms -0,066 ± 0,007 mmol/l; $p<0,05$). A comparison of transportation intensity between extracellular calcium and bone sectors in the main group with the circadian rhythm of blood pressure - night-peakers, depending on the level of blood lipids, showed its even greater slowing down (-0,033±0,004 mg/l; $p<0,05$) in patients with high (above average) levels of total cholesterol (>6.00 mmol/L) and LDL-C (> 3.80 mmol/l) compared with patients in this group, who had lower levels of plasma cholesterol (-0,051 ± 0,005 mg/l).

CONCLUSION

Thus, lipid metabolism disorders are one of the common pathogenetic mechanisms of OA and EH. Dyslipidemia activates system and local inflammation, contributes to endothelial dysfunction, which on the one hand contributes to progression of EG, and on the other hand - through direct destruction of the articular cartilage damage, and indirectly through the subchondral bone damage (resorption, demineralization) leads to more severe manifestations of OA articular syndrome.

FUTURE WORK

Exploring opportunities to improve the efficacy of antihypertensive therapy and treatment of articular syndrome in hypertensive patients with OA by the correction of lipid metabolism.

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SIGNIFICANCE OF DYSLIPIDEMIA IN PATHOGENESIS OF OSTEOARTHRITIS WITH CONCOMITANT ESSENTIAL HYPERTENSION

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Summary. *Purpose. Study influence of dyslipidemia on manifestation of joint syndrome and diurnal blood pressure (BP) profile in patients with osteoarthritis (OA) and concomitant essential hypertension (EH). Subject and methods. Blood lipids, interleukin-1 content, tumor necrosis factor-α), C-reactive protein, Leken's index, C-terminal telopeptide, calcium transport between skeleton and blood, endothelium function, diurnal BP profile were determined in 120 patients with OA and concomitant EH and in 60 patients with EH (comparison groop). Spinal radiographic densitometry was performed. Results. In patients with OA in combination with EH in a greater degree ($p<0.05$), then in comparison group were expressed dyslipidemia, endothelial dysfunction, elevation of proinflammatory markers, bone resorption, deceleration of calcium transport between extracellular and bone sectors and impairment of diurnal BP profile. Moderate correlation was determined between levels of low-density lipoprotein (LDL) cholesterol and degree of endothelial dysfunction ($r=0.543$; $p<0.05$), intensity of calcium transport ($r=-0.557$; $p<0.05$) and Leken's index ($r=+0.551$; $p<0.05$). The most considerable impairments of calcium transport were in patients with «night-peaker» diurnal BP profile and high level of LDL cholesterol (> 3.80 mmol/l). Conclusion. Dyslipidemia is one of the common pathogenetic mechanisms of OA and EH.*

***Key words*:** osteoarthritis, essential hypertension, dyslipidemia, endothelial dysfunction, proinflammatory markers, bone resorption, diurnal blood pressure profile.

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